not by factor IX (which is). I think it fair to call these "defects" of the test in terms of monitoring the patient on oral anticoagulant therapy, but I conclude, with Dr. Quick, that it is the procedure of choice.

I am also in agreement with Dr. Quick as to the usefulness of the prothrombin consumption test to detect platelet dysfunction, although I prefer a modification of his technique for this purpose.

We do a specific assay for factor II (prothrombin) in plasma and serum. Abnormal consumption of prothrombin is confirmed to be caused by a platelet defect when normal results are obtained in a duplicate tube in which blood has clotted in the presence of an optimal amount of cephalin, a platelet substitute.

Dr. Sahud's discussion of platelet function tests concerned newer knowledge about platelet plug formation and did not discuss in any detail the other role of platelets in hemostasis, the provision of phospholipid (platelet factor 3) for coagulation. Dr. Sahud did point out that platelet factor 3 availability could be impaired in qualitative platelet defects. Dr. Quick is quite correct in pointing out that a test for platelet factor 3 is a necessary part of a study of qualitative platelet activity.

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To the Editor: Dr. Quick states that the prothrombin consumption test is sensitive for the detection of the platelet clotting factor. We have performed the prothrombin consumption test on all cases of platelet dysfunction. In the three cases of macrothrombopathia we have studied, the prothrombin consumption test was abnormal as well as other tests of platelet factor 3 availability. However, in five of seven patients with normal size platelets who have a primary platelet disorder as previously described,[1] prothrombin consumption (as measured by a standard method[2]) was normal whereas platelet factor 3 availability by the kaolin method[3] was clearly abnormal.

It may be that the prothrombin consumption test measures a different aspect of platelet procoagulant activity than the kaolin method. In any event, the prothrombin consumption test in the platelet disorders with defective collagen-induced aggregation and normal platelet size has been less sensitive in our hands.

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1. Differential diagnosis of platelet dysfunction (Medical Staff Conference, Sahud MA, Chief Discussant). Calif Med 112:66, Mar 1970
2. Cartwright GE: Diagnostic Laboratory Hematology, Fourth Edition, New York City, Grune and Stratton, 1968, p 380
3. Hardisty RM, Hutton RA: Kaolin clotting time of platelet-rich plasma: Test of platelet factor 3 availability. Brit J Haemat 11:258-268, 1965

Fads, Facts, Fundamentals

To the Editor: Whoever wrote the editorial about "Costly Myths in Medicine" [Calif Med 112:81-82, Mar 1970], said exactly what I have felt needed to be said for a long, long time. Let's have more of the same instead of the completely unopposed and discussed idiotic ideas of current fads and fancies with a complete neglect of facts and fundamentals.

How about a column where some of us could write in and point out some of these things once in awhile?

CHRISTOPHER A. MASON, M.D.
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How about *this* column?—EDITOR

Amniocentesis Registry

To the Editor: Recently there have been two editorials, one in the March 12 issue of *New England Journal of Medicine* by Dr. John Littlefield, and the other in the February issue of *Archives of Environmental Health* by Dr. Robert Cooke, as well as a Medical Progress article in the February issue of CALIFORNIA MEDICINE on recent advances in intrauterine diagnosis for chromosomal and metabolic disorders. There are a large number of such inborn errors of metabolism which can be diagnosed by amniocentesis. Each requires